aorta. Baseline measurements of MDV were obtained on each beagle prior to 2 hours occlusion of the left anterior descending artery (LAD) and then repeated at 4 hours reperfusion and also at day 4, 9, 16, 23 and 30 post. At day 30 post, the heart was removed following MDV measurement and scanned ex-vivo at high spatial resolution before it was sliced and stained with TTC.

**Results:** MDV in the apical region of the heart increased from a mean baseline value of 0.4 ± 0.1 ml/g to 0.9 ± 0.05 ml/g immediately after reperfusion and remained above 0.6 ml/g until day 4 post. From day 9 post MDV normalized except for a thin subendocardial rim in the apical region. High resolution ex-vivo scan of the removed heart confirmed the location of the rim enhancement seen in the MDV image to be subendocardial. TTC staining further confirmed that the rim enhancement was infarcted tissue. MDV in normal myocardium at the lateral free wall of the left ventricle was 0.4 ± 0.05 ml/g at all time points.

**Conclusion:** We concluded from our preliminary results that ECG- gated contrast-enhanced CT scanning is a promising and simple approach for studying myocardial damage from ischemia and its resolution with time.

9:45 a.m.

869-6 **Can Anatomic No-Reflow Be Prevented by Pharmacologic Treatment With Adenosine and Verapamil?**

Thorsten Reffelmann, Robert A. Kloner, The Heart Institute, Good Samaritan Hospital, University of Southern California, Los Angeles, CA

**Objectives:** The aim was to investigate the effects of intravenous adenosine and verapamil on anatomic no-reflow (ANR) in a rabbit model of coronary artery occlusion and reperfusion.

**Background:** Verapamil and adenosine have been successfully used in treatment of clinical no-reflow after direct angioplasty for acute myocardial infarction. However, whether these agents reduce anatomic no-reflow associated with myocardial necrosis, as it occurs in animal models of coronary occlusion and reperfusion, is unknown.

**Methods:** ANR (thioflavin S at end of reperfusion), regional myocardial blood flow (RMBF, radioactive microspheres), and infarct size (IS, triphenyltetrazolium) were compared in anesthetized, open-chest rabbits (ischemia-reperfusion: 30-120 minutes) receiving intravenous adenosine at reperfusion (875 µg/kg bolus, then 175 µg/kg/min until the end of reperfusion) against controls (saline) (n=8, each), and rabbits treated with intravenous verapamil at reperfusion (50 µg/kg bolus, then 150 µg/kg/h) against saline (n=8, each).

**Results:** Both regimes significantly lowered systolic and diastolic blood pressure, reduced specific vascular resistance in the risk area (RA)(adenosine: -38.7% and -38.7% at 30 and 120 min of reperfusion, verapamil: -52.5% and -54.3% at 30 and 120 min of reperfusion), and verapamil increased RMBF within the risk area (verapamil: 48±7%, saline: 33±5% of non-ischemic flow at 120 min of reperfusion). IS (adenosine: 34.1±4.3%, saline: 39.6±6.2% of RA) and ANR (adenosine: 28.8±3.2%, saline: 35.2±5.8% of RA) were not significantly different in the adenosine protocol, and significantly correlated with each other (r=0.98). Similarly, verapamil did not result in significant effects on IS (verapamil: 42.2±5.9%, saline: 38±6.3% of RA), and ANR (verapamil: 37.6±5.5%, saline: 34.9±5.7% of RA), which again showed a significant correlation (r=0.96). ANCOVA analysis revealed that neither treatment uncoupled ANR from IS.

**Conclusion:** Despite reducing vascular resistance within the RA, both adenosine and verapamil at reperfusion did not reduce ANR, suggesting that vasospasm is not a major contributor to anatomic perfusion defects in this experimental model.

ORAL CONTRIBUTIONS

## 872 The Old and the New: The ECG and Novel Biomarkers in Acute Coronary Syndromes

Wednesday, April 02, 2003, 8:30 a.m.-10:00 a.m.
McCormick Place, Room S404

8:30 a.m.

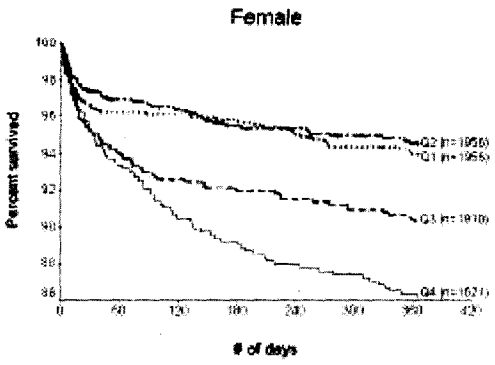
872-1 **Does the Presence of Electrocardiographic Left Ventricular Hypertrophy Predict One-Year Mortality in Non-ST Elevation Acute Coronary Syndromes?**

Michael S. Lauer, Yuling Fu, Wei-Ching Chang, Robert M. Califf, Maarten Simoons, Lars Wallentin, Eric J. Topol, Paul W. Armstrong, Cleveland Clinic Foundation, Cleveland, OH, University of Alberta, Edmonton, Canada

**Background:** Whereas electrocardiographic (ECG) left ventricular hypertrophy (LVH) predicts long-term mortality in otherwise healthy people, its importance as a predictor of death in the setting of NSTEMI acute coronary syndromes (ACS) is not known.

**Methods:** Patients with ACS who were enrolled in the GUSTO IV randomized trial had baseline ECGs read in a blinded core laboratory. LV mass was assessed by Cornell voltage, which is the sum of the amplitude of the S wave in V3 and the R wave in lead V1. LVH was defined as a voltage ≥ 28 mm in men and ≥ 20 mm in women.

**Results:** Baseline ECG data were available in 7443 (95%) of 7800 patients enrolled. ECG LVH was present in 747 patients (10%). During follow-up, 260 patients (3.5%) died by 30 days and 574 (7.7%) by 1 year. ECG LVH tended to predict death at 30 days (4.4% vs. 3.4%, P=0.14), while it was associated with death at one year (12.2% vs. 7.2%, odds ratio = 1.76, 95% CI 1.41 to 2.26, P < 0.0001). There was increased risk of death with increasing Cornell voltage, especially in women. (Figure)




After adjusting for age, ST-segment depression, troponin, C reactive protein, and other confounders using logistic regression, Cornell voltage remained independently predictive of 1-year mortality (p=0.018), but only in women (p=0.031 for the interaction between Cornell voltage and sex; in gender specific analyses, p=0.005 and 0.339 for Cornell voltage in women and men, respectively).

**Conclusion:** ECG LVH at baseline is an independent predictor of 1year mortality among women presenting with ACS.

8:45 a.m.

872-2 **The Prognostic Value of ST-Segment Elevation in Lead AVR in Patients With a First Acute Myocardial Infarction Without Other ST Elevation**

José A. Barrabés Jaume Figueras, Cristina Moure, Josefa Cortadellas, Jordi Soler-Soler, Hospital Universitari Vall d'Hebron, Barcelona, Spain

Background: ST segment elevation (STE) in lead aVR has been associated with severe coronary lesions in patients with acute coronary syndromes, but the prognostic significance of this finding is unknown.

Methods: We analyzed the initial ECG in 775 consecutive patients admitted to our center with a first acute myocardial infarction without STE in leads other than aVR.

Results: Compared to the remaining patients, those with STE in lead aVR had a higher baseline risk profile and a more frequent and extensive ST segment depression in other leads. The rates of death and other in-hospital complications were strongly associated with the magnitude of STE in lead aVR, while CK-MB levels were not (Table). After adjustment for age, Killip class and presence and location of ST segment depression, the odds ratios for death in the last two groups shown in the table were, respectively, 5.6 (95% confidence interval, 2.0-15.5) and 7.8 (3.1-19.9). Among 437 patients catheterized within six months, those with STE in lead aVR had a lower left ventricular ejection fraction and a more extensive coronary artery disease.

Conclusion: Lead aVR contains important prognostic information in patients with a first acute myocardial infarction without other STE. As the worse outcome predicted by STE in lead aVR appears to be related to a more severe coronary artery disease, an early invasive approach might be especially beneficial in these patients.

| | No STE in lead aVR (n=525) | STE 0.05-0.1 mV in lead aVR (n=116) | STE >=0.1 mV in lead aVR (n=134) | P value |
|---|---|---|---|---|
| Death, % | 1.3 | 8.6 | 19.4 | <0.001 |
| Reinfarction, % | 2.1 | 6.0 | 6.0 | 0.009 |
| Angina, % | 10.3 | 19.8 | 26.9 | <0.001 |
| Heart failure, % | 3.2 | 10.3 | 30.6 | <0.001 |
| CK-MB peak, IU/l | 127±103 | 128±102 | 119±95 | NS |
| Left ventricular ejection fraction, % | 66±12 | 63±11 | 59±16 | <0.001 |
| Left main or three vessel disease, % | 22.0 | 42.6 | 66.3 | <0.001 |